and also free nuclei and cells lying in the interstices of the tissue, whose origin is obscure. Emigrated blood-globules are always found. All observers note a thickening of the vessel walls. And from these new formations a process of degeneration in the normal tissue is noticed in the ganglion cells and nerve fibres, as evidence of which are found fat drops, fat crystals, corpora amylacea, and nucleated cells. According to Charcot and Schultze, the nerve fibres lose their medullary sheath while the axis cylinder is not destroyed, but is either swollen or atrophied. The preservation of the axis cylinder explains the absence of secondary degenerations. Other observers have, however, disputed this assertion. The majority of authors agree that multiple sclerosis is a chronic inflammatory process, beginning either in the neuroglia or in the vessel walls.

After this review of previous writers the author proceeds to describe three cases which he has examined by aid of the new staining methods of Weigert and French.

The foci were not sharply limited, but the process faded off into the normal tissue imperceptibly, and was therefore diffuse. In the white substance the basis substance consisted of fine fibrils sharply curved, which were evidently thickened interstitial connective tissue. The vessels were greatly thickened, were tortuous, and were distended with blood. Throughout the foci bodies of shining appearance were found, not connected with the neuroglia fibres, but often lying in the interspaces, and about the same diameter as the axis cylinder. There were naked axis cylinders. No true cellular elements were to be found, nor were Deiter's cells at all prominent. On the periphery of the foci nerve fibres with very thick medullary sheaths were noticed. Near the nerve roots long fibrils of neuroglia were observed running parallel with the nerve fibres, and swollen axis cylinders were also found. In the foci in the gray substance the same network of basis substance and the same naked axis cylinders were seen. The ganglion cells were not affected.

In longitudinal sections it was evident that the basis substance consisted of long straight fibrils, and that the shining bodies were swollen axis cylinders. The axis cylinders were thicker and more swollen in the centre of the focus than at its borders. The medullary substance had at points collected in drops, leaving the cylinder bare at parts between. The cylinders were rarely broken. Such a preservation of the axis cylinders, and such swelling of the cylinders in the midst of sclerotic tissue is found only in multiple disseminated sclerosis, and must be considered characteristic of the lesion. In what relation it stands to the slight degree of paralysis present, the intentional tremor, the excessive reflex action and the other symptoms is undetermined.

The Histological Changes in Spinal Sclerosis. BABINSKI. *Arch. de Physiol. Normal et pathologique*, 1885, p. 186.

In an exhaustive article on this subject the author reviews the microscopic appearances found in disseminated sclerosis, and contrasts them sharply with those observed in secondary sclerosis. He finds that in disseminated sclerosis the degeneration resembles that occurring in the central end of a divided nerve. There is an entire disappearance of the myelin sheath in the plaque, but many axis cylinders remain intact, lying free in the sclerotic tissue. The connective tissue is much increased, and is peculiar on account of the tortuous appearance of the fibrillæ, of which the plaque is composed. The thickening of the vessel walls is intense. In secondary sclerosis the degeneration resembles that occurring in the peripheral end of a divided nerve. The myelin sheaths are not destroyed wholly, many nerve fibres with their sheaths remaining. There are no free axis cylinders to be found. The axis cylinder and its sheath perish together or are preserved together. The connective tissue is increased, but does not consist of twisted strands of fibrils. The changes in the vessel walls are slight in degree but constant.

The author advances the opinion that the sclerosis occurring in locomotor ataxia resembles in its histological characters that occurring in disseminated sclerosis more closely than that occurring in secondary degeneration. He has probably been led to this conclusion chiefly by the analogy already mentioned between the changes occurring in the two ends of a divided nerve and the two forms of sclerosis. This analogy, however, is misleading; for according to the law of Wallerian degeneration a division of the central end of a posterior nerve root between the posterior ganglion and the cord is followed by a centripetal degeneration traceable into the cord, similar in character to the degeneration occurring in the peripheral end of a mixed nerve which has been divided in an extremity. The analogy would therefore indicate that the changes in a locomotor ataxia should resemble those in the peripheral portion of the divided nerve, which are similar, as the author shows, to those occurring in secondary degeneration of the cord. No one who is familiar with the appearance of disseminated sclerotic plaques and with the changes in locomotor ataxia could confound the two, as the absence of the twisted and coeild fibrils of connective tissue in the latter lesion is very noticeable. On the other hand a comparison of the sclerosis in locomotor ataxia in the posterior root zone with the sclerosis in the posterior median column (which is admitted by all authorities to be an ascending degeneration secondary to the lesion in the posterior root zone), shows that the histological changes are very closely allied, if not identical. In both there are an increase of connective tissue, a thickening of the vessel walls, and an obliteration of many nerve fibres, the entire fibre, axis cylinder, and myelin sheath being destroyed together; but nowhere are naked axis cylinders preserved and myelin sheaths alone destroyed.

M. A. STARR.